United Journal of Veterinary Science and Technology

Short Communication

Keel-Bone Damage in Laying Hens-Can We Prevent It?

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Volume 1 Issue 1- 2018

Received Date: 10 Oct 2018

Accepted Date: 26 Oct 2018

Published Date: 03 Nov 2018

1. Short Comunaction

During the last decade, the pressure on farmers has increased, especially considering the animal welfare. In the poultryindustry, the pressure of animal activists, public and food industry has led to the banning of the conventional cages for use in laying hen production by European Union in January 2012. Instead, use of enriched cages or free-range rearing has been enforced. However, transition to this kind of laying hen rearing has led to unexpectedly high incidence of keel bone fractures [1], resulting in pain, changes in behaviour [2], and consequently overall decrease of their welfare [3].

There are several mutually interacting factors causing the keel bone fractures in laying hens. These include the age of the birds, nutrition, housing conditions and genetic susceptibility.

When assessing the incidence of keel bone damage (whether it is a fracture or a deviation of the bone), it is important to take into account the age of the animals, as it was proven that its prevalence, as well as its severity, increase through the laying period [4, 5]. Furthermore, it has been proven that perches have a causal role for some types of keel bone fractures, as reviewed by [3]. On the other hand, removing of the perches from bird's environment would have a negative impact on their welfare because they provide a crucial resource for roosting, which is a part of hen's natural behaviour. Few studies have shown, however, that appropriate design of the perches can influence the occurrence of keel bone damages [6, 7].

It was confirmed that calcium shortage could be one of the causes of the high incidence of keel bone fractures in laying hens. Due to selection, today's genotypes of laying hens can lay up to 300 eggs compared to only 20 per year for red jungle fowl. It takes a lot of calcium to form an eggshell each day, which is mobilised partly from hen's bone. This calcium has to be restored each day, and this can be done by adding the calcium with larger particle sizes during the day, such as grit or shells, which will then be digested during the night [8]. In addition, it has been documented that omega-3 fatty acids influence bone strength [9]. Hens utilise foraging diet with equal n3 and n6 omega fatty acids ratio, and typical commercial feed has a 6 to 10 fold excess of omega 6-fatty acids.

A number of studies have reported that occurrence of keel bone damage and its quality varies among different strains [7, 10, 11], thus breeding of the hens resistant to fracture presents a potential solution for this welfare problem. Furthermore, the investigations showed that heritability of bone mineral density, a major determinant of bone quality, ranges from 0.39 to 0.59 [12, 13]. This gives a good basis for the potential use of genomic solutions to the problem of keel bone fracture in laying hens. Indeed, [14], found somatostatin receptor 5 (SSTR5; NM_001024834), calcium channel, voltage-dependent, T type, alpha 1H subunit (CACNA1H; ENSGALG00000005215), mitochondrial ribosomal protein S18A (MRPS18A), and glycoprotein

1b, alpha polypeptide (GP1BA; ENSGALG00000021693) eQTLs to be connected with bone min-

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Citation: Kusec ID, Keel-Bone Damage in Laying Hens-Can We Prevent It?. United Journal of Veterinary Science and Technology. 2018; 1(1): 1-2.

eral density in poultry, but also in mouse and human. on the other hand, [15,16]. Reported a new locus associated with femur bone mineral density and nine SNPs in genes that were associated with bone quality. Three of them (RANKL, ADAMTS and SOST) are already known to be associated with osteoporosis in humans. The connection of all these SNPs to similar conditions in other species makes them thus good candidate genes for this condition in laying hens. However, their potential role in keel bone fracture remains to be confirmed.

In conclusion, keel bone fracture in hens represents a big issue, not only from the animal welfare standpoint but also for the egg industry worldwide. Most definitely, the solution lies in solving all the mutually interacting issues rather than solving only one or some of them.

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